

## Acute myocardial infarction without obstructive coronary artery disease demonstrated by selective cinearteriography

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*Among 118 patients who had had a well-documented acute myocardial infarction and who were studied by selective coronary arteriography, in 5 cases (4.2%) no obstructive lesions of the coronary arteries could be detected. The left ventricular angiogram showed an abnormal contraction pattern in 3 instances. The electrocardiogram and vectorcardiogram showed infarct patterns in 3 cases and transient disturbances of repolarization in 2. Of the latter, one patient developed an infarction with classical electrocardiographic picture 10 months later. A second coronary arteriogram was made which showed a total obstruction of the anterior descending artery. Gradually the electrocardiographic alterations vanished; a third coronary arteriogram made after another 18 months revealed partial recanalization of the obstructed artery. It seems that a primary thrombo-embolic process could be responsible for the disease pattern. After the first coronary arteriogram appeared normal, anticoagulant therapy was discontinued; in retrospect the very normality of the arteriogram in conjunction with the typical clinical picture of myocardial ischaemia probably should have constituted an indication for permanent anticoagulant therapy. In this case the serum enzymes never reached pathological levels. In all the other cases the enzymes showed characteristic increases. One of these cases was that of a 33-year-old woman who was 36 weeks pregnant when the infarction occurred. A spontaneous and uncomplicated delivery of a healthy child followed 3 weeks later. Coronary angiography revealed an aneurysm of the anterior descending artery without narrowing. It is possible that the aneurysm had originated as a dissecting aneurysm which temporarily occluded the lumen. In the remaining 3 cases no specific features suggesting an aetiological explanation were found. Some possible underlying causes are discussed.*

Cases in which the absence of obstructive lesions in the coronary arteriogram seem to contradict an indisputable clinical picture of coronary artery disease have been reported in practically all studies dealing with the relation between coronary angiogram and clinical findings. Generally in these cases there is a history of chronic occurrence of typical angina pectoris or there is electrocardiographic evidence, at rest or after exercise, of myocardial ischaemia, or both. Patients, however, presenting the clinical picture of acute myocardial infarction in whom subsequent coronary

arteriography failed to demonstrate any obstruction to account for the clinical symptoms have only rarely been described. Campeau *et al.* (1968) published 6 such cases, 2 of them having a Starr-Edwards prosthesis, and one with aortic stenosis in whom the infarction had occurred during retrograde left heart catheterization. Eliot and Bratt (1969) pointed to the occurrence of myocardial necrosis in young women with apparently normal coronary arteries, which was confirmed by necropsy in 3 instances. A case report of a 19-year-old youth was presented by Sidd, Kemp, and Gorlin (1970), and 3 cases were briefly mentioned by Ross and Friesinger (1966). In the material of Proudft, Shirey, and Sones (1966) the coronary arteriogram was normal in 13 out of the 50 cases listed as 'possible myocardial infarction'

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and in 37 of the 174 patients classified as 'coronary failure'. However, in the paper no clinical details are reported.

In our material of 900 patients who underwent selective coronary arteriography there were 118 patients who had had a well-documented acute myocardial infarction; in 5 of them no obstructive lesions could be demonstrated. In one patient, however, a second coronary arteriogram made 8 months later revealed a total occlusion of a major branch, and in another case an aneurysm of the anterior descending artery was noted. It is the purpose of this paper to present the diagnostic features and to discuss the aetiology of these cases.

## Methods

Selective coronary arteriography was performed according to Sones' technique. The 5 in field of a Philips 5"/9" image intensifier linked with a cine-pulse unit was employed for the recording of the coronary arteriogram; the left ventricular angiogram was filmed using the 9 in field. Recordings were made on 35 mm cine-film with a speed of 60 frames a second. The contrast medium was Urografin 70 or 76 per cent. In every case both coronary arteries were filmed in multiple left and right anterior oblique projections; the recordings of the left ventricular angiogram were made in the right anterior oblique projection.

The electrocardiogram was taken in the usual way including 6 limb leads and 8 praecordial leads.

Routinely two vectorcardiographic systems (Frank and Burger) were employed. The vectorcardiograph which was used possesses a set of 3 oscilloscopes for simultaneous photographic recording of the 3 orthogonal vector loops (frontal, horizontal, and left sagittal). In addition, the X, Y, and Z components of the vectorcardiogram were recorded by means of a high frequency ink-jet recorder.

The upper limits of normal of the cardiac enzymes are by our laboratory standards as follows: serum aspartate aminotransferase (SGOT) 25 U/ml; serum creatine phosphokinase (SCPK) 30 mU/ml; and serum lactic dehydrogenase (SLDH) 350 U/ml. The SLDH fractions were determined semiquantitatively (cardiac isoenzymes fractions 1 and 2).

## Case reports

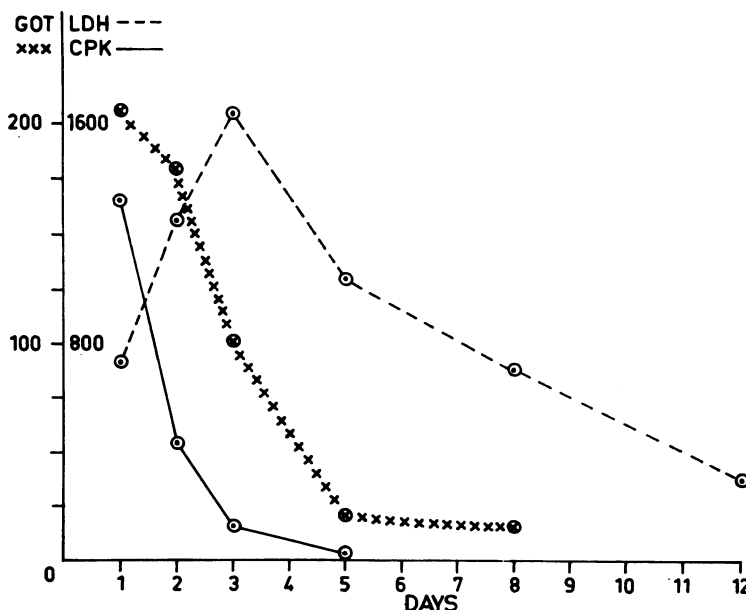
**Case 1** A 33-year-old woman, who was 36 weeks pregnant, was admitted to the cardiology department in September 1968. Up to the day of her admission she had enjoyed good health; two previous pregnancies had been entirely uncomplicated. During her housework in the morning she was surprised by an attack of severe retrosternal pain radiating into the neck, the shoulders, and both arms. On admission, approximately 3 hours later, she was still in pain. Physical examination revealed crepitations over the basal lung fields and a third heart sound of moderate intensity;

otherwise there were no signs of cardiac failure. No cardiac murmurs were heard. The blood pressure was 105/85 mmHg. The laboratory findings (Fig. 1) were characteristic of extensive myocardial necrosis. The electrocardiogram (Fig. 2a) and the vectorcardiogram (Fig. 2b) showed the development of an anterior wall infarction. The patient was treated with digoxin and bed-rest. Because of her pregnancy, no anticoagulants were administered. A spontaneous and uncomplicated delivery of a healthy child followed three weeks later. She left the hospital 34 days after admission in apparently good physical condition. After discharge she remained virtually asymptomatic.

In February 1969 she was readmitted for coronary arteriography. On admission a grade 1/6 systolic murmur was heard in the 4th left intercostal space. The chest x-ray showed a slightly enlarged cardiac silhouette. The coronary arteriogram revealed a small aneurysm, but no narrowing, at the origin of the anterior descending branch of the left coronary artery (Fig. 3); otherwise all major branches were normal. The left ventricular angiogram showed conspicuously increased end-diastolic and end-systolic volumes, the contraction pattern being synergic. Up to now the clinical picture has remained essentially unchanged.

**Case 2** A 31-year-old man, who had sporadically experienced nocturnal chest pains during the past 5 years, was admitted to a hospital elsewhere in March 1968, because of severe and prolonged retrosternal pain. An electrocardiogram which was taken on admission showed ST elevations

FIG. 1 Case 1. Characteristic increase in serum enzyme levels with typical course during the days after the acute attack.



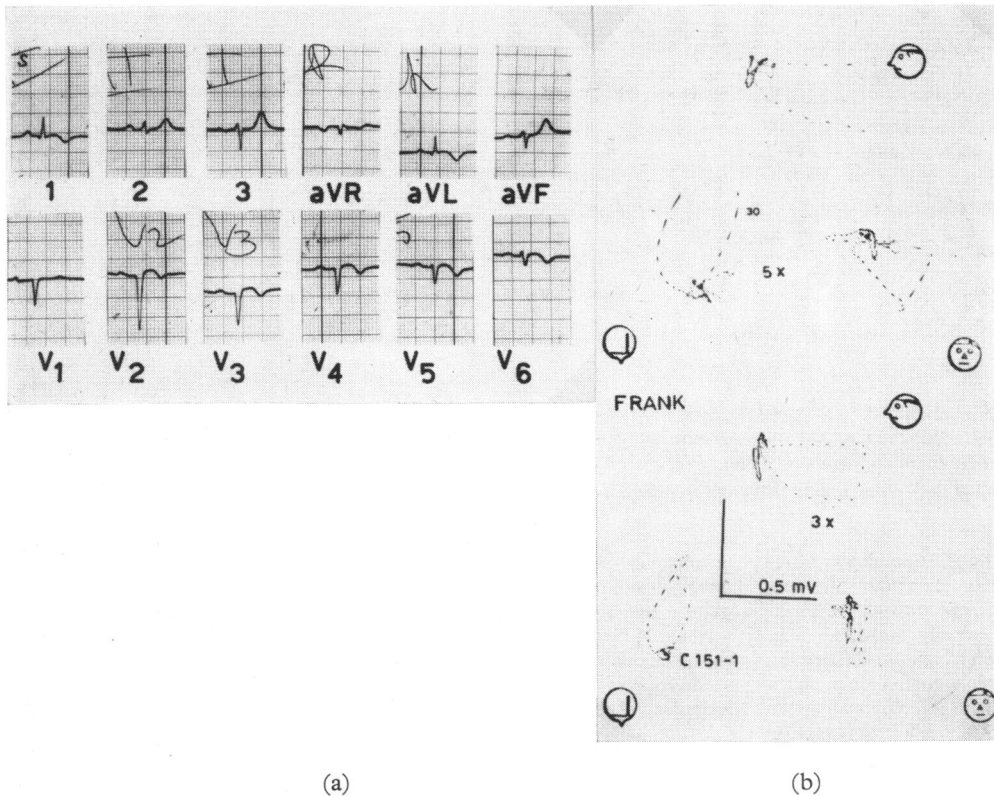
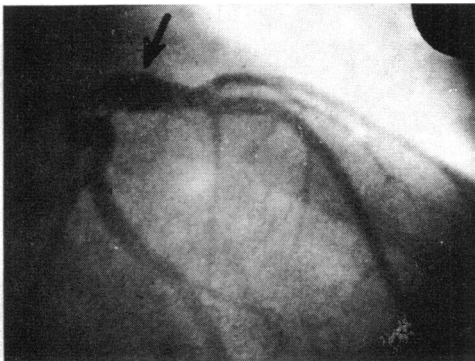


FIG. 2 Case 1. (a) Electrocardiogram taken 10 days after admission; picture of recent anterior wall infarction. (b) Vectorcardiogram recorded at same time. Anterolateral and high lateral defects (centrifugal limb is directed sharply downward and shows a sudden posterior deviation after 10 msec). Acute repolarization disturbance (displacement of the T point to the left and anteriorly; T loop deformed and T axis almost perpendicularly downward). The time marks are at 2.5 msec intervals and form arrows in the direction of the inscription of the loops.

FIG. 3 Case 1. Single frame from cine-arteriogram showing the left coronary artery in the right anterior oblique view. The arrow points to a fusiform aneurysm of the anterior descending artery; no narrowings.



over the entire anterior wall (Fig. 4a). The serum enzymes remained within normal limits. The patient was treated with bed-rest and anticoagulants. The electrocardiogram became completely normal within a few days, but transient ST elevations were noted again after mobilization. After discharge his chief complaint consisted of nocturnal chest pains; his electrocardiogram and vectorcardiogram remained entirely normal.

The patient was referred to us for coronary arteriography which was performed in May 1968. The coronary arteriogram showed no abnormalities (Fig. 5a), the left ventricular angiogram being also normal. Anticoagulant therapy was discontinued. During the next 5 months the patient was virtually asymptomatic, but in October he again noted chest pains, progressively increasing both in frequency and severity. Prolonged and severe attacks necessitated readmission to the hospital in January 1969. Serial serum enzyme determinations again yielded normal results. The electrocardiogram and vectorcardiogram, which were still practically normal on admission, showed a very slowly developing extensive anterior wall

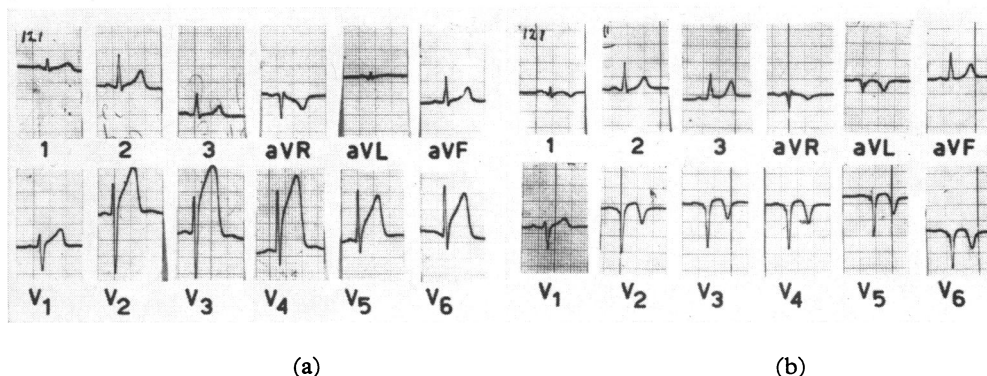


FIG. 4 Case 2. (a) Electrocardiogram recorded at first admission elsewhere (March 1968). Acute repolarization disturbance over entire anterior wall which disappeared completely within a few days. (b) Electrocardiogram in January 1969. Picture of extensive anterior wall infarction which developed slowly over a few days after admission for chest pains.

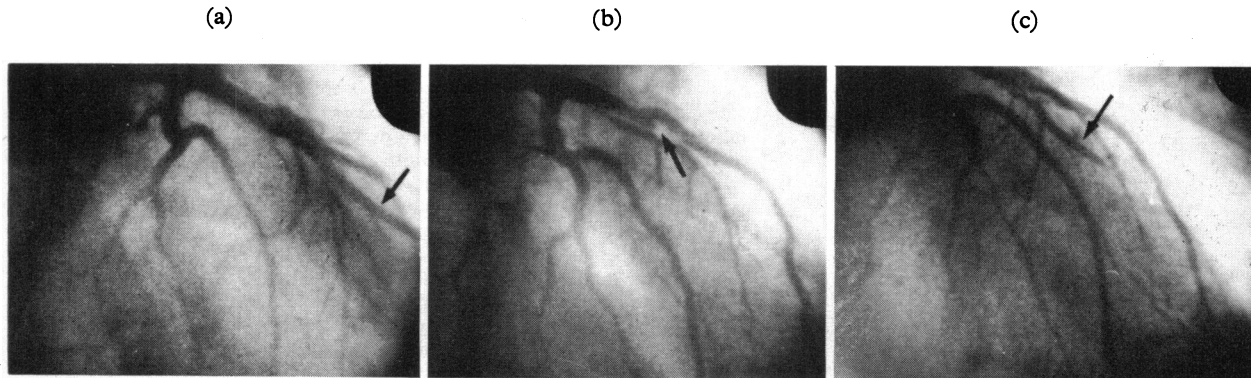
infarction (Fig. 4b and 6a). Four weeks after admission a second coronary arteriography was performed. On this occasion the anterior descending artery appeared to be totally occluded (Fig. 5b). Anticoagulant therapy was reinstituted. After discharge the patient reported only slight anginal complaints, and the electrocardiogram and vectorcardiogram showed a gradual reversion to the previously observed normal picture (Fig. 6b).

In July 1970 his condition rapidly deteriorated, typical and severe attacks of angina being provoked by the slightest exertion. The patient was again admitted to the hospital. The serum enzyme levels remained normal throughout and his electrocardiogram unchanged (the only abnormality being a residual 40 msec Q wave in lead aVL). In August 1970 a third coronary arterio-

graphy was performed. The anterior descending artery now appeared to be patent over approximately two-thirds of its length (Fig. 5c). There was some, but not severe, narrowing at the site of the previous occlusion. The filling with contrast medium was delayed with respect to the other branches which were still normal.

**Case 3** A man, 43 years of age, was admitted because of retrosternal pain of moderate intensity radiating to the left shoulder. Physical examination revealed no abnormalities. The electrocardiogram showed conspicuously elevated ST segments in leads V<sub>4</sub>–V<sub>6</sub>; a few days later the T waves became isoelectric, and afterwards diphasic. Approximately 14 days after admission the cardiogram had become normal again. The first day of admission the serum enzymes were high (Table), the levels

FIG. 5 Case 2. Single frames from cinearteriograms showing the left coronary artery in right anterior oblique projections at nearly the same degree of obliquity. (a) May 1968, no abnormalities; note normal anterior descending branch (arrow). (b) February 1969, anterior descending artery occluded at its origin (arrow). (c) August 1970, the anterior descending branch (arrow) is again partially opacified. The filling with contrast medium is delayed. Subsequent frames show that the artery is patent over approximately two-thirds of its length. There is only a slight narrowing at the site of the previous occlusion.



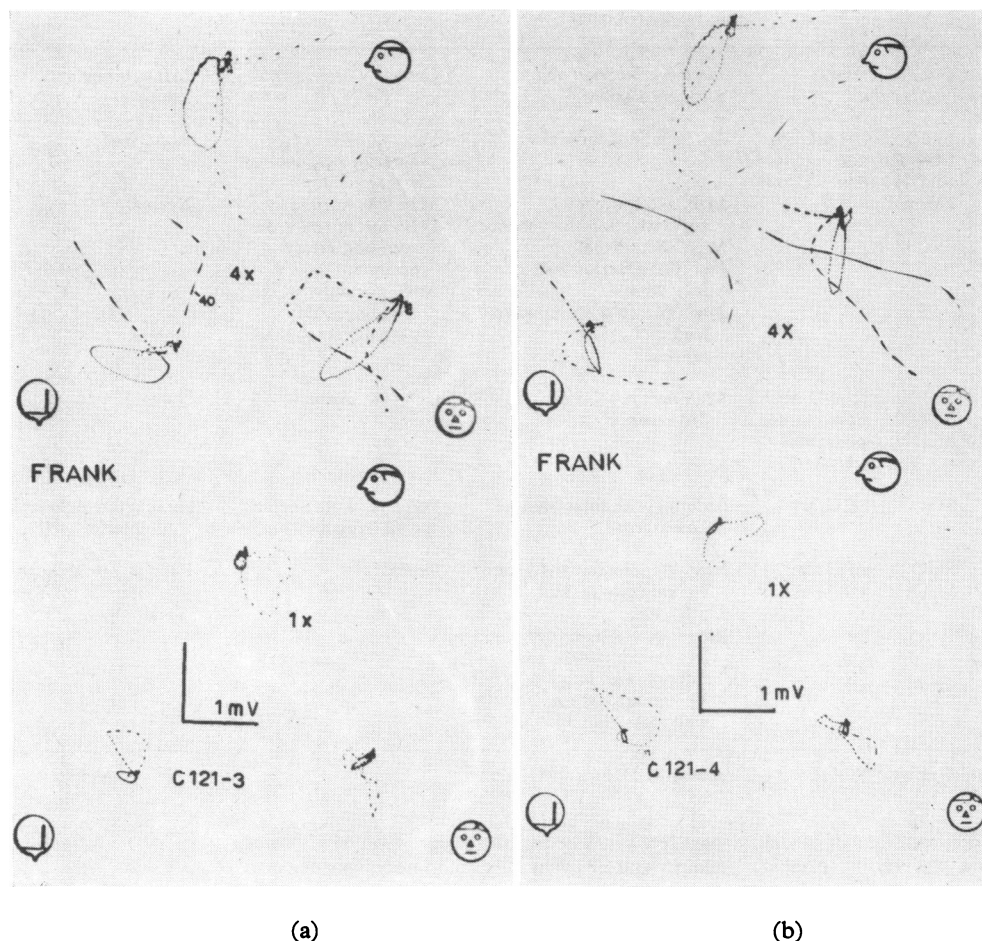


FIG. 6 Case 2. (a) Vectorcardiogram recorded at the same time as electrocardiogram of Fig. 4 (January 1969). Anterolateral and high lateral defects (centrifugal limb is oriented inferiorly and to the right, turns sharply posteriorly after 25 msec and does not reach to the left). Severe disturbance of repolarization pointing to a subacute anterior and anterolateral infarction (large symmetrical T loop with its axis inferior and to the right; anterior displacement of J point). (b) Vectorcardiogram (May 1969) reverting to the normal picture observed before January 1969. The leftward activity of the QRS loop has been largely restored. Repolarization is still moderately abnormal (T axis too much inferior and to the right; clockwise inscription of horizontal T loop; slight anterior displacement of J point).

reverting to normal within 3 days. Coronary arteriography, which was performed 4 weeks after admission, revealed no abnormalities. The left ventricular angiogram was also normal.

**Case 4** A 43-year-old man had, since October 1965, been under care elsewhere where he was admitted with severe chest pain of acute onset. The electrocardiogram showed the characteristic development of an anteroseptal infarction. During the first days an intermittent right bundle-branch block and ventricular extrasystoles were also noted. The SGOT rose to 174 U. The patient was given anticoagulants and corticosteroids. After discharge from the hospital his chief complaint

consisted of exertional dyspnoea, and in addition he suffered from chest pains which gradually acquired a typically anginal character. Attacks of severe nocturnal dyspnoea necessitated readmissions to the hospital in February 1966 and November 1967. The patient was on digitalis and diuretics.

Because he was thought to have severe coronary artery disease, he was referred to us for coronary arteriography and, if possible, coronary surgery. While on the waiting list, he experienced, in January 1970, an attack of chest pain which was not relieved by nitroglycerin. A new acute infarction was suspected and the patient was subsequently admitted to our coronary care unit.

TABLE *Case histories*

<i>Patient's sex and age (yr)</i>	<i>Date of infarction</i>	<i>Cardiac enzymes (peak values)</i>	<i>Electrocardiogram and vectorcardiogram</i>	<i>Coronary arteriogram</i>	<i>Left ventricular angiogram</i>
F 33 (36 weeks pregnant)	Sept. '68	SCP: 1320 mU/ml SGOT: 206 U/ml SLDH: 1620 U/ml	Anterior wall infarction	Feb. '69: aneurysm anterior descending artery	Abnormal
M 31	March '68(?) Jan. '69	Normal	March '68: obvious repolarization disturbance May '68: normal Jan. '69: anterior wall infarction Aug. '70: 40 msec Q wave in lead aVL, otherwise normal	May '68: normal Feb. '69: anterior descending artery occluded Aug. '70: anterior descending artery partially recanalized	Normal
M 43	Sept. '68	SCP: 170 mU/ml SGOT: 57 U/ml SLDH: isoenzymes 1 and 2 elevated	Transient repolarization disturbance	Oct. '68: normal	Normal
M 43	Oct. '65	SGOT: 174 U/ml	Anterior wall infarction (aneurysm?)	Feb. '70: slight vessel wall irregularities	Paradoxical pulsations of anterior wall
M 34	Nov. '65	SGOT*: 193 U/ml SLDH*: 328 U/ml	Nov. '65: electrocardiogram: typical inferior wall infarction June '70: electrocardiogram: left axis deviation and inferior wall ischaemia Vectorcardiogram: inferior wall infarction	Normal	Lack of movement of portion of inferior wall

\* Determined elsewhere, normal values: SGOT < 45 U/ml and SLDH < 180 U/ml.

Physical examination disclosed no signs of cardiac failure. The blood pressure was 100/80 mmHg. On auscultation of the heart a third sound was heard; no murmurs were noted. The cardiac enzyme levels remained normal. The electrocardiogram showed a stationary pattern of anteroseptal infarction. Several vectorcardiograms were taken, all showing the same pattern of anterior and high lateral defects with intraventricular conduction delay and a severely disturbed repolarization: this picture was considered to be indicative of a left ventricular aneurysm (Fig. 7). Coronary arteriography, performed 3 weeks after admission, revealed only insignificant vessel wall irregularities of the proximal portions of the right coronary artery and anterior descending branch of the left. The left ventricular angiogram (Fig. 8a) disclosed much enlarged diastolic and end-systolic volumes and paradoxical pulsations of a large portion of the anterior wall.

**Case 5** The patient was first admitted to a hospital elsewhere in November 1965, because an acute myocardial infarction was suspected. He was then 29 years old. Serial electrocardiograms (Fig. 9) taken at that time showed an inferior wall infarction with a classical progression. The cardiac enzymes which were determined on the day of admission and on the next day were high (Table). The patient received anticoagulants. After discharge from the hospital he reported retrosternal pains, usually 15 minutes in duration, occurring both after exercise and spontaneously. Over the

past few years he noted a progression of his complaints and he eventually could no longer perform his work as a gardener.

In June 1970, the patient was admitted to our department for coronary arteriography. The electrocardiogram no longer showed an infarction pattern, though there were still signs of inferior wall ischaemia. The vectorcardiogram, on the other hand, showed a distinct inferior wall defect (Fig. 10). The coronary arteries appeared to be entirely normal; the left ventricular angiogram, however, showed absence of contractions of a portion of the inferior wall adjoining the mitral annulus (Fig. 8b).

## Discussion

The case histories of the 5 patients are summarized in the Table. They represent 4.2 per cent of the total number of patients who had a previous history of acute myocardial infarction and who were studied by coronary arteriography. It may be remarked that a few patients were encountered who presented with typical chest pain, but in whom the electrocardiographic or enzymatic changes were not sufficiently convincing; these were not included in this study.

All 5 patients here described were quite young (29 to 43 years old) when the infarction occurred. They were all normotensive and all

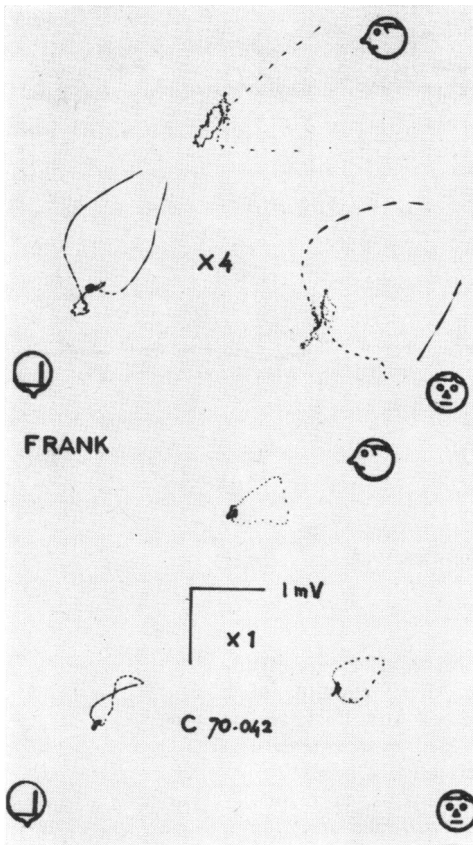


FIG. 7 Case 4. Vectorcardiogram of February 1970. Extensive defect of anterior and lateral walls with impairment of intra-ventricular conduction. Obvious anomaly of repolarization (T axis to the right anterior and inferior; clockwise inscription of horizontal T loop; conspicuous anterior displacement of J point). Picture characteristic of left ventricular aneurysm.

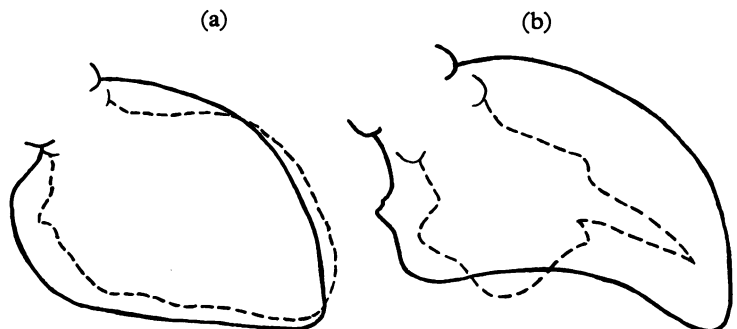
had normal serum cholesterol and beta-lipoprotein levels and normal glucose tolerance tests.

The first patient was pregnant, which is remarkable, since the occurrence of myocardial infarction during pregnancy appears to be a rarity. Reviewing the published material up to 1960, Watson *et al.* (1960) were able to collect only 22 cases, among them one of their own. In 1967, 7 other cases (among them 2 cases from the published reports) were added by Fletcher, Knox, and Morton. If 4 cases reported by Mendelson (1960) are also included, the number of published cases of myocardial infarction during pregnancy totals 33. Coronary atherosclerosis or syphilitic

ostial stenosis were usually considered to be the underlying disorders, but in one case a large thrombus in the right coronary artery was found whereas the vessels appeared normal (Muir, 1960). In our patient a thromboembolic occlusion followed by recanalization might have occurred. On the other hand, one is inclined to speculate about some relation between the episode of myocardial necrosis and the aneurysm of the anterior descending artery demonstrated by coronary arteriography. In this context it is of interest to note that of a total of 20 reported cases of isolated dissecting aneurysm of the anterior descending artery, 6 occurred in the postpartum period (Nishiyama *et al.*, 1967). We wonder whether the aneurysm of our patient had started as a dissecting aneurysm which temporarily occluded the lumen. If this hypothesis were true, the patient would represent the first published case in which the diagnosis had been established during life. This would mean that dissection of the anterior descending artery does not necessarily have a fatal outcome.

The second case also shows particularly interesting elements. The initial, transient electrocardiographic disturbances of repolarization incriminated the anterior wall as the compromised region. The first coronary arteriogram which was entirely normal seemed to repudiate this. However, 8 months

FIG. 8 Left ventriculograms; superimposed outlines of left ventricular areas at end diastole (solid lines) and end systole (broken lines), in the right anterior oblique view. (a) (Case 4) much enlarged maximal diastolic and end-systolic volumes; generalized decreased contractions; outward bulging of a large portion of the anterior wall during systole. (b) (Case 5) a portion of the diaphragmatic wall adjoining the mitral annulus shows lack of contraction and even suggests paradoxical movement.





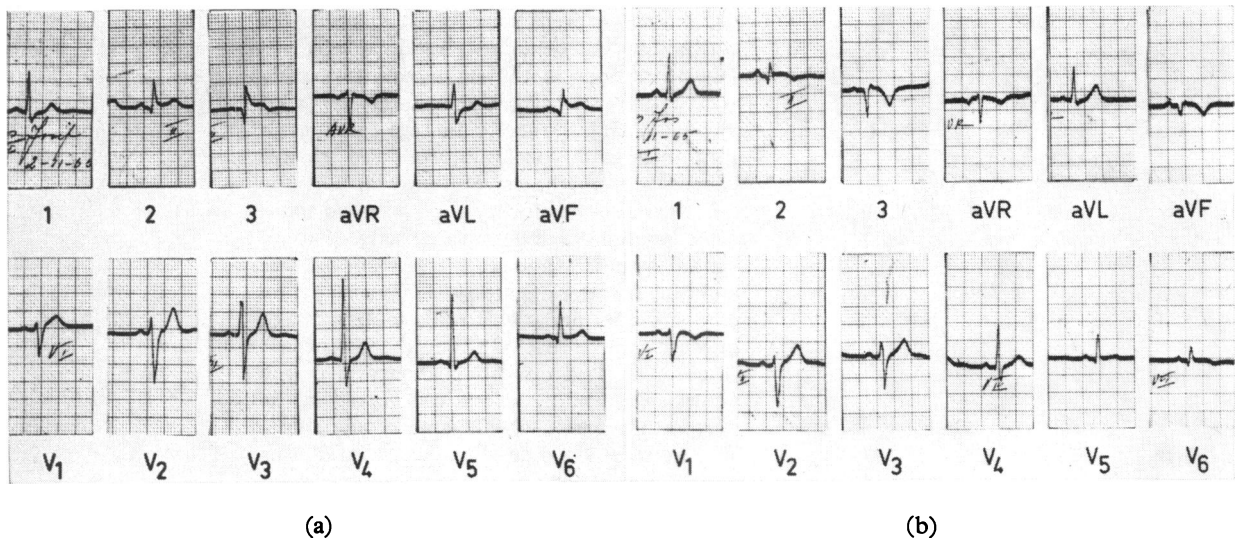


FIG. 9 Case 5. Electrocardiogram in November 1965. (a) on the day of admission and, (b) 3 days later, illustrating the classical course of a diaphragmatic wall infarction.

later the anterior descending artery was found to be totally occluded at the second arteriography. Now the electrocardiogram and vectorcardiogram evidenced defects of the anterior and anterolateral walls. Quite astonishingly, these abnormalities gradually vanished again to yield the previously observed normal electrocardiographic patterns. A third coronary arteriogram revealed partial recanalization of the occluded artery. The several features of this case point towards a primary thromboembolic process as the possible aetiology. By this assumption the normality of the first coronary arteriogram can be explained either by lysis of previously present thrombi or by the possibility that the obstructions were still limited to the microcirculation. The unusually slow progression of the electrocardiological infarction pattern without accompanying increases in the cardiac enzymes, as was observed during his second admission, also appears to be in agreement with the supposition of a thrombus with progressive centripetal spread. The partial recanalization and the absence of an important narrowing at the site of the previous occlusion as observed at the third coronary arteriography corroborate this reconstruction. According to Himbert *et al.* (1963), 'primitive' coronary thrombosis involves exclusively the anterior descending artery. The authors observed 3 such cases. At necropsy the arteries appeared to be free from atherosclerosis; however, other slight nonobstructive changes of the vessel wall were noted in all these cases. It is conceivable that such changes, which

probably escape recognition by arteriography, provide a substrate for the formation of platelet aggregates (Mustard and Packham, 1969) and subsequently lead to progressive thrombosis. Unfortunately such a possibility was not fully appreciated by us, and anticoagulant therapy was discontinued after the first coronary arteriogram appeared to be normal. While the efficaciousness of anticoagulants in atherosclerotic heart disease may still be a matter of dispute, we feel at present that the very absence of atherosclerotic narrowings, despite a clinically convincing picture of acute myocardial infarction or ischaemia, should raise the suspicion of a thromboembolic process, and indeed may be regarded as a stringent indication for anticoagulant therapy. In our case consideration should also be given to the possibility that a traumatic lesion of the vessel wall had been produced at the first coronary arteriography. However, since the procedure was followed by an interval of 5 months during which the patient was virtually asymptomatic and of 8 months during which the electrocardiogram remained normal, this hypothesis does not stand up.

In the other 3 patients here described there were no specific characteristics leading up to an aetiological explanation. Also in these cases reversible thromboembolic processes are among the possible causes, but no evidence as to this cause can be brought forward. Other factors having been mentioned as possible causes of myocardial ischaemia and necrosis include carbon monoxide intoxication, ab-



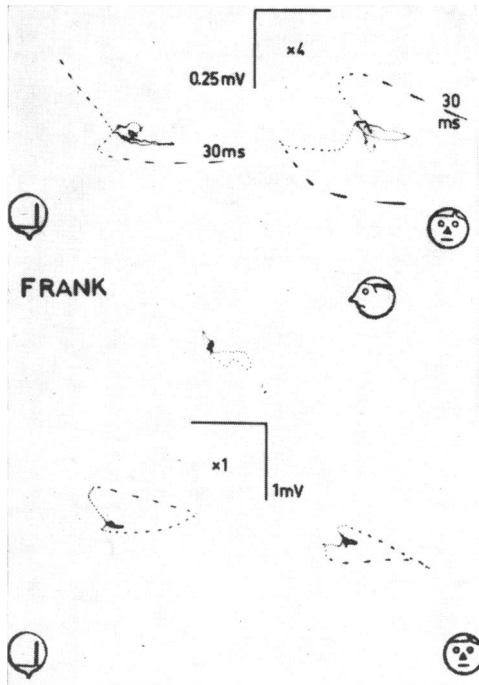


FIG. 10 Case 5. Vectorcardiogram of June 1970. Old inferior wall defect (centrifugal limb remains superiorly located for more than 30 msec, being deviated to the left at the same time).

normal haemoglobin-oxygen dissociation (Likoff, Segal, and Kasparian, 1967; Eliot and Bratt, 1969) and small vessel disease (James, 1969). However, in our patients carbon monoxide poisoning could practically be excluded, and it is difficult to understand how the other causes could result in myocardial infarction of such acute onset as was observed in our cases.

One may also suppose that an occlusion (e.g. at the origin of a small branch) has been overlooked, but particularly in the cases in which laboratory findings and electrocardiographic changes indicated extensive myocardial necrosis, or the left ventricular angiogram showed a grossly abnormal contraction pattern whereas the major coronary arteries undoubtedly were intact, this explanation is not plausible.

Consideration should be given to the possibility that some of our cases actually represent cases of cardiomyopathy or myocarditis. This problem deserves particular attention in Case 1, since some relation between cardiomyopathy and pregnancy has been suggested (Becker and Taube, 1962; Brown *et al.*, 1967; Mendelson, 1960; Walsh *et al.*, 1965; Hughes *et al.*, 1970), though most

authors consider only cardiomyopathy occurring in the postpartum period as belonging to this supposed entity. However, while cardiomyopathy and myocarditis may be associated with pain suggesting myocardial infarction, the typical development of electrocardiographic infarction patterns in conjunction with enzymatic alterations is unlikely to occur in these diseases.

In conclusion, it is shown again by these 5 cases that myocardial infarction is not necessarily related to permanent major coronary artery disease. Presumably processes other than coronary atherosclerosis are involved more often than is generally expected. At present the nature of these disorders is still obscure; nor is it known whether a single cause (e.g. primary coronary thrombosis) or a variety of processes is involved. Further research in this field is of paramount importance for a complete understanding of so-called ischaemic heart disease.

Our sincere thanks are due to Drs J. de Geus, Utrecht, Th. F. Fickenscher, Bilthoven, and A. E. V. Hillebrand, Tegelen, for making available to us their records of Cases 3, 4, and 5, respectively.

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